treatment. The author believes that during this time the intestine had suffered changes sufficient to account for the duration of the symptoms. He concludes: "One is fully justified in assuming that in these cases balantidia were the cause of the diarrhœa, and that the main indication in the way of treatment is their destruction."

In all of these cases the treatment advised by Waldenstroem and Henschen, namely, large enemata (2500 c.c.), to which 50 to 75 c.c. vinegar and 5 to 7.5 tannin were added, resulted in a rapid disappearance of the parasites.

Recent observations appear to have settled beyond a doubt the question as to the pathogenicity of this parasite.

STRONG and MUSGRAVE (*Bulletin of Johns Hopkins Hospital*, 1901, vol. xii., p. 31) report from the Philippines a case of diarrhœa resulting fatally in four months, in which the stools contained great numbers of balantidium coli. The blood showed an increase in the number of eosinophilic cells. At necropsy the lower part of the jejunum and ileum were reddened and contained considerable mucus. In the large intestine the mucosa throughout was reddened and covered with bloody mucus; there were also a number of shallow ulcers. Balantidia were demonstrated throughout the mucosa and passing through the submucosa and muscularis.

A most elaborate study of this question is that of SOLOWJEW (*Centralblatt f. Bakt.*, 1901, vol. xxix., pp. 821, 849), who has also demonstrated that balantidium coli may enter into the healthy mucosa between the glands. "Making their way further into the submucosa they increase markedly and bring about there also extensive changes. Hence, they penetrate the muscularis mucosæ between the muscle bundles. The most marked changes being observed in the submucosa, the necrosis begins here. This spreads in all directions, reaching on the one side the muscular coat, on the other the mucosa. The glands deprived in this spot of their proper nourishment become necrotic if they have not already been destroyed by the parasites.

"Thus, the general opinion that balantidium coli is met with only on the surface of the mucosa, where it feeds upon the mucus, cannot be considered correct. The presence of the parasites in the depths of the tissues explains the malady produced by them, as well as the tendency to frequent relapses after their apparent complete disappearance."

This article is accompanied by a full table of references.

[There would appear to be little specific in the clinical manifestations of these cases which, in some instances, present the symptoms of a chronic diarrhœa, in others of an acute dysentery, with mucus and bloody dejecta. The reviewer has seen some of Strong's specimens which are convincing as to the pathogenicity of the parasite.—W. S. T.]

The Etiology of Acute Dysentery in the United States.—Under the direction of FLEXNER, VEDDER and DUVAL (*Journal of Experimental Medicine*, 1902, vol. vi., No. 2, p. 181) have undertaken to solve two problems: 1. To determine by comparative study whether the organisms described by Shiga in Japan, by Flexner and Strong in the Philippines, and by Kruse in Germany are of the same species, and 2, to discover the cause of acute dysentery in the United States, and if possible to identify it with the organisms of the observers mentioned. In other words, to determine whether acute dysentery is the same the world over, and whether it be due to a specific organism, bacillus dysenteriæ Shiga.

After briefly describing their technique they state that before the organism under consideration can be considered to be bacillus dysenteriæ, it must have fulfilled the following requirements:

a. It must give the proper cultural characteristics, as shown by standard cultures of Shiga, Flexner, and Kruse.

b. It must possess the right morphology, as shown by the same.

c. It must give a positive agglutinative reaction with the same of the known dysenteric sera.

Their material was drawn from various sources. The bacillus dysenteriæ was grown from the stools of five different cases studied in various Philadelphia hospitals. It was also obtained in three cases of dysentery which occurred in the Lancaster County Almshouse and Insane Asylum, where several deaths from the disease had occurred. They also had the opportunity of studying a severe epidemic of dysentery which broke out in the Springside Home, New Haven, Conn. In this epidemic more than fifty cases occurred within three weeks among the 350 inmates, all being of a very acute form, with fatalities. They obtained the bacillus in fourteen of these cases which they studied. Three of these were insane patients.

They describe in detail the cultural characteristics, morphology, and agglutination reactions of the organism. The agglutination tests were made with the organisms obtained from Shiga, Flexner, Kruse, and Strong and with the organisms which they isolated from their own patients. The tests were made with the blood of the patients they observed, and also with a sample of antidysenteric serum sent by Shiga to Flexner. Briefly stated, the agglutination reaction was practically identical with all the organisms.

As a result of their study of the cases in this country, they firmly believe that there is a specific bacillus, and that it is identical with the organism described by the observers mentioned. They have no doubt that the organism is pathogenic. It is interesting to note, however, that in no instance have characteristic intestinal lesions followed inoculation of animals with the organism. Flexner and Strong have both produced typical dysentery in man following the ingestion of bacillus dysenteriæ.

It is important to note that the organism in this country has been found both in sporadic and epidemic cases; also that it is apparently the cause of acute dysentery in the insane. It is also present in the cases of "terminal" dysentery in association with chronic Bright's disease.

They draw the following conclusions from their study:

1. The several standard cultures used in this study are indistinguishable—a conclusion previously reached and stated by Flexner.

2. The acute dysentery of the United States is due to a bacillus indistinguishable from that obtained from the epidemics of dysentery in other parts of the world.

3. The sporadic and the institutional outbreaks of acute dysentery are caused by some micro-organism, and this organism is identical with that causing epidemic acute dysentery.

4. The cause of acute dysentery, whether sporadic, institutional, or epidemic, is bacillus dysenteriæ Shiga.